ON INSUFFICIENCY OF THE AORTIC VALVES

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ON

INSUFFICIENCY

OF THE

AORTIC VALVES,

IN CONNECTION WITH SUDDEN DEATH;

WITH NOTES, HISTORICAL AND CRITICAL.

BY

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PREFACE.

The first issue of this Essay, 1861, has been some time exhausted. In this re-issue, such additions have been made as seemed necessary for a statement sufficiently inclusive of the points involved.

The historic portion will, it is hoped, be found tolerably full and accurate. With regard to the second portion, during the nineteen years that have elapsed, I venture to doubt whether any addition of real importance has been made to the pathology of the disease, excepting, perhaps, the influence of strain upon the valves, so ably illustrated by Dr. Clifford Allbutt. Symptoms, doubtless, have been more minutely studied; physical phenomena more accurately estimated by instrumental means; and the intermittent murmur of Duroziez found, in doubtful cases, of great confirmatory value.

Research into the history of pathology authorises the belief that, in the past, the disease must have existed as an occasional, though unrecognised, cause of sudden death—or a cause, at best, but very dimly discerned until little more than a century ago. Ex-

perience, later and larger, has shown the comparative frequency with which the disease occurs, followed, at times, by the result in question.

For practical ends, the disease may be said to present itself chiefly under two forms. Such distinction, however, must not be thought to have more than a simply relative value.

In the one form, the compensating hypertrophy fails comparatively early, either from some original or acquired infirmity of cardiac fibre, or from its fatty degeneration, and life, if not abruptly closed, ends by gradual asthenia. In the other form, the compensative adjustment is so perfect, and the fibre inherently of so excellent a quality, as to permit long years of active life to pass uncompromised by cardiac failure.

4, Suffolk Place, Pall Mall,

March 30*th*, 1880.

ON

INSUFFICIENCY OF THE AORTIC VALVES,

ETC., ETC.

Part I.

HISTORIC LITERATURE.

'Nescire quid antea quam natus sis, acciderit, id est semper esse puerum.'—Cicero.

It is proposed, in the present sketch, to trace, from the time of Harvey, the successive steps by which our own refined knowledge of the disease in question has been attained. Such an outline ought not to be without interest, at least, for those more immediately concerned in this branch of pathology. It does not, however, appear that much inquiry has been made, of late, in this direction.

It is clear that insufficiency of the aortic valves could find no place in pathology, prior to the discovery of the circulation of the blood. A correct appreciation of the mechanism of this process, and of the methods of physical diagnosis, are the indispensable pre-requisites for a scientific knowledge of the diseases of the heart. Without adequate ideas of the former, not even a centralization of the signs of such diseases was possible; and, in default of the latter, no differential diagnosis

was attainable.* But no sooner was the circulation of the blood a truth of demonstration, than physiological pathology soon achieved the richest conquests in cardiac diseases.

* At a period in the history of cardiac science, long centuries remote from that indicated, *Galen* had already suspected that certain irregularities of the circulation might be attributable to obstruction occurring at the orifices of the heart. He thus describes the case of *Antipater*, a physician of some eminence practising at *Rome*. 'When between fifty and sixty years of age, Antipater was attacked by the ephemeral fever, on the cessation of which, he examined his own pulse with the view to further treatment. Detecting great irregularity in the movement of the arteries, he became, at first, greatly alarmed; but, soon afterwards, feeling that the fever had entirely left him, he took a bath; his physical power being exhausted by work and want of rest. He next adopted a guarded regimen, till the end of the third day, reckoning from the accession of the fever. Having experienced no relapse, he resumed his ordinary occupation; but he was greatly surprised, on feeling subsequently the same irregularity of the radial pulse. Meeting him one day, he held out his hand, and, laughing, asked me to feel his pulse. I, also, smiling, replied, "What enigma are you propounding?" He again, laughing, begged me to feel his pulse. I found great irregularity in the pulse, not only in that totality of the pulsations named systematic, but also in each single dilatation of the artery. Astonished to see him alive with such a pulse, I asked him if he felt the slightest difficulty in breathing. He answered, "None whatever." I watched each change that occurred, constantly feeling the artery of the wrist, during a period of six months. As he asked me at the commencement of what diathesis I supposed him affected, and how such condition could cause a similar pulse without fever, I replied, that in my Treatise upon the Pulse, I had adverted to a similar irregularity. Indeed, I am of opinion that it results from an obstruction of the great arteries of the lung (mitral valve?). "But," said I to him, "the obstruction could not be the cause of the inflammation of the viscus within you, or, you would have fever. We must only suppose that the obstruction caused

We owe to our distinguished countryman, Mayow, the philosophic exposition of the common law which governs the changes sustained by the heart, consecutive to structural derangements of its valvular apparatus*—the law, as it is now termed, of retro-dilatation and hypertrophy.

It is to be regretted that later authorities should have been so unmindful of the observations of this most original physiologist. He thus describes the pathologic sequence in a case of obstruction of the left auriculo-ventricular orifice† :—

by thick and viscid humours on the formation of a crude tubercle, has produced in you a like diathesis." "But, then," interrupted Antipater, "I should have an asthmatic orthopnœa." "What you state," I replied, "is probable, but still not exact, for such an orthopnœa would equally well be produced by the accumulation of this thick and viscid humour, not in the smooth arteries, but in the trachea." I ordered him to adopt a regimen similar to that recommended in asthmatic cases, and to take medicines having the same properties as those used in such cases. After an interval of six months, he experienced slight dyspnœa and palpitation of the heart; at the onset, only once, then three, four, or even more attacks; the dyspnœa progressively augmenting to about the fifteenth day; then, suddenly, the breathing became extremely laborious, his powers failed, and he died quickly, in a similar way to other individuals suffering from disease of the heart.' In the opinion of Macoppe, *De Aortæ Polypo*, Antipater died from dilatation of the aorta. (*Vide* 'Lectures by the Author, upon The Historic Literature of the Pathology of the Heart and Great Vessels, Part i, from the earliest authentic records, to the close of the Arabian epoch.' 1860, page 25.)

* Mayow's work bears date, Oxon., 1669. Some have attributed the views taken by Mayow to Blancard; but, according to both Morgagni and Haller, Blancard was frequently guilty of the grossest plagiarism.

† Mayow acknowledges himself indebted to his friend, T. Millington, for the particulars of the case.

With respect to insufficiency of the aortic valves, the first clearly described case that I have met with is by one of the greatest masters of cardiac pathology of the seventeenth century—Raimond Vieussens.

In his introductory remarks to this case, Vieussens observes: 'I am aware that some anatomists have found the trunk of the aorta ossified, both in men and animals; but I have never either read or heard of changes of structure of the sigmoid valves of this vessel.* I have, nevertheless, once found these valves ossified, as is shown from the following observations:— A man, aged thirty, of melancholic temperament, subject for a long period to attacks of epilepsy, was

by the pulmonary artery; they, consequently, the right ventricle as well as the right auricle, and perhaps, also, the caval veins, undergo successive dilatation. Generally, the walls of dilated hearts are softened, although at the same time often thickened, not merely with respect to their cellular tissue, but as to their actual sarcous element. See Vieussens, *Traité du Cœur*, ch. 16, 1715, for a similar chain of reasoning. Also Treviranus, *Biologie*, Band iv, p. 231.

* It is singular that the same remark should have been made by Lancisi, the great contemporary of Vieussens. He does not appear, however, to have had an insight into the pathology of aortic insufficiency. The following passage is from his work, *De Subitaneis Mortibus*, ed. 1706, p. 100, s. vii: —'Igitur, ad posterioris animadversionem transeuntes, ingenue fatemur, hoc sarcomatum vitium in tot observatis cadaveribus nunquàm alias nobis fuisse compertum, hoc adhuc ex aliorum lectione cognitum; utcumque viderimus hujusmodi valvulas cartilagineas, osseas, exesas, phlogosi affectas, aut cum sanguiferis varicosis.' It is, however, quite certain that diseases of the valves had been described by several authors before either Vieusseus or Lancisi. *Vide Morgagni*, Letter 23, a. 10.

seized, in the course of the year 1695, with so violent a paroxysm, as to cause alarm lest some vessel should rupture in the brain. However, on being brought to the hospital, the attack so soon abated, that, if not quite convalescent, he was, at all events, out of immediate danger. Nevertheless, on my visit to the hospital, I examined the case. His expression was dull; face, pale and bloated; pulse very full, very frequent, very hard, somewhat unequal, and so strong that the arteries in both arms conveyed to my finger the sensation of a tense and strongly vibrating cord. A pulse of this kind, such as I have never before observed, and trust never again to encounter, impressed me with the conviction that the patient must suffer from violent palpitation of the heart. This opinion proved correct; for, upon questioning him, he informed me that for some considerable time he could neither lay upon the sides or back, unless the head were greatly elevated, being prevented by the violent palpitation of the heart. He added, moreover, that when he tried to lie upon either side, especially upon the left, it seemed to him as if the stroke of a hammer fell upon his ribs. After the examination, I observed to the surrounding physicians and students, that a polypus of considerable size had formed in the left auricle, but that the left ventricle was free. But, in consequence of the absence of dyspnœa, together with the character of the pulse, that the passage of blood from the right to the left heart was free and

unobstructed, which never happened if a polypus of any size had formed in either of the ventricles. I also stated, moreover, that independently of the polypus, there was something unusual in the case, concerning which I could form no accurate opinion, but which the death of the patient would soon unravel. My prognosis was justified, for the patient died in three days after this investigation.

'*Autopsy.*—A polypus was found in the right auricle, the left ventricle was dilated to an extraordinary degree, and the ascending aorta was thickened, hard, and cartilaginous. The sigmoid valves were serrated on their unattached borders, resembling, in shape, the teeth of a saw, and, in consistence, stone or gypsum.

'*Remarks.*—In consequence of the rigid state of the aortic semilunar valves, very powerful action of the left ventricle was necessary to propel the blood into the aorta; and, since these valves were serrated and torn at their free border, they could no longer close: consequently, at each systole of the aorta, blood was forced back into the cavity of the ventricle. There existed, therefore, a derangement of the circulation, produced by the rigidity and calcification of the aortic valves, which had caused the increased action of the heart.'

In the year 1749, Sénac gave to the world his classical monograph upon the diseases of the heart. It

is literally, as has been said of it, '*opus magni sudoris.*'

This work contains the results of a life of almost undivided labour in the cause. Writings which obtained the unqualified commendation of Morgagni and Haller, need no further recommendation to the modern investigator of the history of cardiac pathology.

Sénac was clearly cognisant both of obstructive and regurgitant valvular disease, and of the influence these conditions exercise both upon the chambers of the heart and the arterial pulse. But his knowledge of *obstructive* disease was, apparently, restricted to the arterial sigmoid valves, and that of *regurgitant* disease to the mitral valves.* He mentions, however, two cases; one from a great authority on diseases of the heart, Ballonius; the other from Malpighi, which would seem to be connected with aortic insufficiency, from dilatation of the aortic mouth.

In the case of Ballonius, a man aged twenty-two was long troubled with palpitation. There was visible beating of all the arteries. He died suddenly after swallowing a bole of cassia, which was supposed to

* Tome ii, liv. iv, ch. ix. When the valves of the aorta are ossified, the blood is impeded in its exit; the pulse is small, and the heart liable to palpitation. But, when the auriculo-ventricular valves are ossified, and incapable of closure, as in the case narrated, what derangements disturb the action of the heart? The blood must reflow into the auricles during the contraction of the ventricles. Less blood passes into the aorta, and the pulse, consequently, is but little dilated; but if the auricle is distended at the time the ventricle contracts, palpitation necessarily occurs.

have caused his death. The true cause, however, of this unexpected event, resided in the aorta and heart, the cavities of which were extremely dilated.

The case narrated by Malpighi runs as follows :—

'A man, in whom the pulse was strong and tense, and into whose arteries the blood was projected with violence, had the left ventricle so large that it could have contained a heart of moderate size. The area of the aorta was three inches, and its internal surface thick set with bony scales, and the walls very solid.'

The great author of the Letters on the *Seat and Causes of Diseases*, has devoted several of them to the consideration of the diseases of the heart. With few exceptions, these letters contain all that is known, even now, with regard to cardiac pathology. The reflections that Morgagni has made upon the subject of aortic insufficiency in connection with sudden death, are so judicious that they apply equally at present as when written. The following case is given in the twenty-seventh letter:—

'12. A young man, of an excellent habit and proper conformation of body, and of a good stature, having been long troubled with a difficulty of breathing, was relieved by blood bursting forth, every now and then, from the nostrils. But, after he had begun to be deficient in this discharge, he happened to take a long journey from Trent to Padua, which he performed, partly on foot and partly on horseback, within the space of two days. Immediately after coming off his

journey, and while he was stooping to his portmanteau, which was laid on the ground, he fell down dead.

The thorax being cut into on the day following, the vessels of the head and the neck were previously observed to be very turgid with blood. But when the cavity was opened there appeared to be no extravasation in it, nor were the lungs seen to adhere, anywhere, to the pleura. Both the lobes of this viscus, however, were livid, from the blood stagnating in them; and they were small besides, from the vast magnitude of the heart, which (when the pericardium was cut into, where there was no small quantity of reddish serum) appeared to be even bigger than that of a bullock. And, indeed, the auricles, and the right ventricle, were bigger than usual; yet, the greatest bigness was observed to be in the left ventricle. Nor was this owing to the parietes, which were not thicker than usual; but to the cavity of the ventricle being dilated to such a degree that nothing could be conceived as being more so, and not only filled with a quantity of black blood (that had not formed itself into polypus concretions, though it was somewhat collected into grumous coagula), like the other cavities of the heart, but also distended therewith. Besides these appearances, the semilunar valves, which lie at the orifice of it, were not bony, indeed, but hard; and what immediately occurred to the eyes, very small, for they were contracted and corrugated. But the great artery, although it was not larger than it naturally is, yet in proportion

as it receded from the heart, so much the thinner than usual were its coats. Nor was its internal surface entirely free from longitudinal furrows, notwithstanding they were somewhat obscure. However, in the heart and the whole thorax, there was nothing besides that was not natural. . . . .

'14. You will, perhaps, ask why, as all the four cavities of the heart were dilated in the young man in question, the dilatation of the left ventricle was, nevertheless, the greatest. Without doubt, because the dilatation of this ventricle had given rise to the dilatation of the other ventricle and the two auricles; that is, by admitting a less quantity of blood than it ought, for the reason which I have just now given you; and, in consequence of this obstruction, by retarding the motion of the blood in the left auricle, in the lungs, in the right ventricle and its adjoining auricle.

'But why was the left ventricle the first of all to be dilated? Why? Certainly, because the semilunar valves, whatever the cause of this circumstance might be, having been contracted and corrugated, could not properly expand themselves so as to prevent the blood being, in part, sent back into the ventricle from whence it came, during the contraction of the aorta; which part of the blood would, perhaps, have been less, if the coats of the more distant parts of the aorta had been able to drive on towards the veins the proper quantity of blood which it had received; but this, the thinness of these coats—that is, the decreased number of their fleshy and elastic fibres—did not permit.'

In the 23rd Letter, also, Article 12, he again alludes to obstructive and regurgitant disease of the aortic valves, and shows most clearly how the lungs and right ventricle, at times, become implicated :—

'Wherefore, in the woman also spoken of by me, besides the aorta being here and there bony, or inclining to a bony state, the valves of it are, also, to be attended to. For, as one of these was bony, and the others indurated, so being, of consequence, less yielding to the blood, they might increase the obstacles to its exit, and, on the other hand, not sufficiently prevent its return, when soon after repulsed by the great artery ; so that, as some portion of it returned into the left ventricle of the heart, when the ventricle ought to receive the blood that was coming in from the lungs, it would necessarily happen, that the returning portion, as well as the portion which had not been extruded just before, must occupy some part of that space which, from the design of nature, was entirely due to the blood that was coming in from the lungs ; which circumstance, finally, could not but overload both the lungs and the heart, and compel the latter to throw out, every now and then, with a great impetus, the blood that stagnated in it.'

Christian Theophilus Selle, a physician of celebrity, published in 1790 his *Observations on Medicine*, a portion of which was translated by Brocklesby. That he saw the pathological bearing of the disease, is

clear from the details of his 24th observation, 'Ossification of the Valves of the Aorta'.

'In 1781, a young man of robust constitution, aged seventeen or eighteen, was brought to the Maison de Charité.

'I observed a considerable pulsation in all his arteries. He had violent palpitation of the heart, and a pulse singularly large, quick, and hard, although quite regular. He could get no rest, from the difficulty in lying down.

'All the other functions were in their natural state.

'I had no doubt of organic disease, and reasonably presumed that an aneurism existed, which would, sooner or later, burst and cause death.

'I tried to diminish the mass of the blood by bleeding, but this did not at all lessen the pulsation of the arteries, neither were saline nor anodyne medicines of greater efficacy. A leuco-phlegmatic condition ensued, and death soon followed. Upon opening the body, I was greatly astonished to find no disease in the vessels. All the viscera were equally sound, but the valves of the heart were ossified and immobile. They were greatly retracted, and in such a way, that the blood ejected by the heart could readily pass; but, as a consequence of such retraction, it would naturally reflow, and occasion this disorder of the arterial system.'

John Hunter, 1794, records a very anomalous case of cardiac disease of long duration, in which no

special diagnosis could be made. After death, the aortic valves were found to be diseased and incompetent. The comments upon the case are worthy the fame of the great physiologist.

Allan Burns*, in his admirable work, had clearly enough discerned both the nature and occasionally sudden termination of the affection. 'When the arterial valves are thickened and corrugated, and incapable of covering the orifice which they ought to guard, we find that the symptoms are generally pretty nearly the same with those mentioned in the last case, but in other instances they are not very prominently marked; nay, in some cases, the person dies without any suspicion of being affected with any serious disease. A girl, while employed one morning in the laborious occupation of washing heavy clothes, was seized with a sudden fit of illness; she had just time to turn to her companion, when she fell down dead, by the side of her tub. The aorta was thickened and indurated, and the semilunar valves were rigid and imperfect; each flap was at least the eighth part of an inch thick; all of them were corrugated, and one was so puckered as not to be larger than a split pea.

'In this girl the valves were in such a state, that they were incompetent to close the passage from the ventricle into the aorta. They were so much diseased,

* *Observations on some of the most frequent and important Diseases of the Heart*, etc., p. 189. Edinburgh, 1809.

that they would prevent the free transmission of the blood from the heart into the artery, but at the same time their condition was such, that they would have less influence in this way than in allowing a part of the blood on the contraction of the vessel to return into the ventricle.

'From these two causes there must have been a deficiency of arterial blood in the vascular system. The patient must have had an incapacity to exert herself, as she had been wont to do; but in her, as the disease was on the increase, the circulation was, while tranquil, carried on tolerably well. When, however, she over-exerted herself, as she did the morning before her death, the effects of the altered state of the aortic valves would come to be more seriously felt. From the repercussions of the aorta being now frequent, blood must very often have been driven back into the ventricle. It must have gained on that cavity, producing an irregularity in the action of the heart which would soon induce a state of deep syncope.'

It is stated, though very briefly, by Professor Friedreich, in his excellent work upon *Diseases of the Heart*,* that insufficiency of the aortic valves was known to Stoll, Sénac, and Kreysig.

Although tolerably familiar with the writings of these great masters, I did not remember discovering

* Virchow's *Handbuch*. Fünfter Band, zweite Abtheilung, zweite Hälfte, s. 367.

sufficient evidence of such knowledge, in its strict sense. I have reperused their works, and still incline to my opinion. I may, however, have overlooked the particular passages upon which Professor Friedreich founds his assertion.

Maximilian Stoll, one of the most consummate practitioners of his day, whose penetration so little escaped, is stated to have recorded a case of sudden death from this affection. Such statement, however, is hardly correct. He does, it is quite true, narrate an instance of sudden death where the aortic valves were found diseased. In commenting upon the case, he observes, 'We attributed the sudden death to the increased flow of blood to the heart when (the patient, a woman aged thirty) first mounted the stairs, and, after undressing, got into bed. The blood, by reason of the ossification of the aorta and the rigidity of the valves, was incapable of passing and moving onwards.' But the disease partook greatly more of an obstructive than of a regurgitant character. In his own words,* 'Aortæ principium, ut primum ex corde emergit, paucarum linearum tractu osseum fere totum fuit. Valvulæ ejusdem crassæ, partem cartilagineæ fuere, partim vero ejus naturæ, quæ inter os et cartilaginem ambigit; parum mobiles, claudere, sive aperire aortam opporteret, et semi-hiantes fuere'.

Although the heart was greatly enlarged, there is no

* *Pars Prima Rationis Medendi*, sectio decima-sexta, p. 250. 1777.

mention made of the distension, with blood, of the left ventricle; none of any peculiarity of the pulse or vessels generally; points which so accurate an observer as Stoll would hardly have failed to note, and upon which he would, unquestionably, have laid very particular stress.

Moreover, there was found considerable effusion into each pleural sac, and some effusion into the pericardium. The liver was of immense size (*ingens*); the spleen thrice its natural size; also, marked intestinal congestion existed. The patient had been admitted into the hospital for abdominal disorder.

Stoll simply closes his remarks by pointing out the mischief likely to result from venesection in such cases, where marked rigidity of the vessels exists.

Kreysig mentions a case* where the patient fell suddenly dead at a festive gathering, 'as if struck by lightning'. It is one of the most extraordinary on record so far as regards the *post mortem* results, unless paralleled by that recorded by De Hahen.† Nevertheless, it bears no semblance to one of aortic insufficiency. Further on (s. 586), he describes the various changes occurring in the arterial sigmoid valves, but in the sense of obstruction only, not of insufficiency, although he was acquainted with the case detailed by

* *Die Krankheiten des Herzens,* zweiter Theil, 2te Abtheilung, s. 575. 1816.

† *Ratio Medendi.* Pars II, cap. viii.

Selle. Chapters VII and VIII abound with cases and sage reflections upon the diseases of the several valves. In Article 4, he treats of forcible pulsation of the arteries of the neck, but in no connection with aortic insufficiency.

The more interesting part of Kreysig's writings, as specially connected with the peculiar pulse of aortic insufficiency, occurs as stated below, and is thus expressed.*

'Testa describes a peculiarity of the pulse in cardiac affections which he compares to that of air rushing and whizzing under the finger, and expresses surprise that no previous writer should have mentioned it. He states, moreover, that he observed it in one case associated with violent palpitation of the heart and large arteries. Here, the recovery of the patient proved his suspicion of aneurism to have been unfounded. I have myself, on many occasions, detected this remarkable peculiarity of the pulse, conjoined with excessively violent and irregular action of the heart. At other times, the pulse was not so markedly whizzing, but, so to speak, crepitant (I know no native word which clearly conveys the idea of the sensation imparted to the finger, except it be that of two cartilaginous substances grating against each other).† I am unable to give

* *Op. cit., Erster allgemeiner Theil, über die Pathologie und Diagnostic enthält,* s. 301. Berlin, 1814.

† In some cases of great insufficiency, combined with marked anæmia, the feeling, at times, actually imparted to the finger of the explorer of the pulse, is as if the walls of the artery came rubbing together at the close of the collapse.—J. C.

either an exact physiological or diagnostic explanation of the peculiarity in question. It would seem to me that, in one form, blood was propelled through some narrowed outlet in quantity insufficient to fill the *lumen* of the artery, and therefore, probably, some aeriform element was developed, which more completely distended the arterial wall; the other instance might be explained on the supposition of an abnormally violent tension of the arterial coats engendering in them a sort of tonic contraction. I observed this form of pulse in a case of hypertropy of the left ventricle combined with dilatation of the aorta. We possess, however, a special memoir on the subject by *Mauchart*, who also detected it in aneurism of the aorta.'*

The following is Testa's own description of this form of pulse, at least as nearly as I can render it. 'At other times, both in aneurismal and in purely cardiac cases, a different condition of the pulse is found, one which, although difficult to explain in fitting terms, nevertheless appears to me to convey to the finger specially, a sensation of *bruit*, of thrill, of internal sibilation, as if air were shot most rapidly under the finger when the artery is compressed; this sort of *bruit*, moreover, so far as I have been able to ascertain, is not always perfectly alike, either in respect of the tension, or of the sensation which it

* Kreysig. *Die Krankheiten des Herzens. Erster allgemeiner Theil*, s. 301. Berlin, 1814.

generates. Nevertheless, this sensation of internal suspiration, resembling a little puff of air, remaining but an instant, appears to differ from the pulse which, too, has been termed 'murmurish' or 'crepitant', and which approaches more nearly than any other to the pulse termed 'dicrotic', with which it is, at times, united, and which some distinguished authorities of the French School have classified under the term 'rebounding.*

There exists, as is evident, a most striking analogy in the description of this form of pulse, as furnished by these two great observers.

But, from these *excerpta,* it can hardly be maintained that either *Stoll, Kreysig,* or *Testa* recognised aortic insufficiency in any other than an accidental light, and not, as a rule, connected, sequentially, with sudden death. Indeed, the extent of their knowledge, in this particular, was behind that of some of their great predecessors.

But, it is abundantly clear that both Testa and Kreysig had discerned (though without knowledge of its true cause) the main characteristics of this pulse of many names — thrilling — jerking — splashing — hammering — collapsing — names derived from the special sensation communicated to the finger of the explorer in the particular case. Perhaps, the sen-

* A. G. Testa. *Delle Mallatie del Cuore,* vol. 2do, p. 203. Napoli, 1841.

sation imparted, as of air being rapidly shot under the finger at times, is as accurate a description as some of the terms in current use.

It is especial matter of regret that the classical writings of Testa and Kreysig have not been made more accessible to us by translation, enriching as they do so greatly the science of cardiac pathology. At the present time, in devoting so much labour and attention to physical refinements, we must take heed lest the *Science* be lost sight of in the *Art*.

The writings of Laennec show no familiarity with the disease in question. Although well acquainted with the changes of the aortic sigmoid valves, which obstruct the onward current of the blood,* he does not appear to have been aware that these might, also, permit a regurgitant one. It is the more singular that the marvellous sagacity of Laennec should have failed him here, inasmuch as he has even shown how the valves were, at times, curled round upon themselves.†

It may, perhaps, be matter of question, whether he did not occasionally confound aortic insufficiency with what he designated 'Spasm of the Arteries, with murmur and fremitus'.‡ His distinguished editor,

* *Traité de l'Auscultation*. 1837.

† *Op. cit.*, tome iii, p. 264-9.

‡ *Op. cit.*, p. 513-15. In a large number of cases, in which slightly marked bellows murmur exists in some arteries, the radial pulse conveys a peculiar fremitus, a kind of vibration exactly re-

Andral, gives him no credit for knowledge of the affection, but admits it to be of later growth.*

Bertin, whose collected writings upon the *Diseases of the Heart* were edited by Bouillaud, and published in 1824, has devoted a most valuable section to the diseases of the aorta and sigmoid valves, consequent upon inflammation.

This distinguished pathologist, however, was so completely fettered by the dogma of *Frank*, respecting the common occurrence of arteritis, that he has actually (obs. xxvii) detailed a case of aortic insufficiency, from dilatation of the mouth of the aorta, and overlooked it, in consequence of preconceived views. It is headed: —'*Thinning, with Dilatation of the Walls of the left Ventricle: Inflammation and Ossification of the Aorta, etc.* Anne Berger, huxtress, ætat. forty-seven, had suffered for about two years from spasms and other anomalous symptoms, which were supposed to be of a nervous character, dependent upon the "critical period". She was treated by various practitioners, who all regarded her complaint as "nervous", and prescribed accordingly calmatives and anti-spasmo-

sembling that of a tense metallic cord, which, after being made to vibrate, is touched with the tip of the finger. This character of the pulse is, probably, that observed by Corvisart in cases of ossification of the mitral valve where fremitus exists over the cardiac region. It would appear to be a simple diminution of this latter phenomenon . . . . [This view of Laennec appears to be similar to that of Bertin.—J. C.]

* *Op. cit.*, tome iii, p. 283.

dics, but without success. On her admission into the Hospital Cochin, the eighteenth prairial, an. 11 :— She complained of great dyspnœa. Each moment she was in dread of suffocation, and obliged to keep constantly in a sitting position ; she felt, she stated, as if something rose in her throat and threatened to choke her. She complained of a sensation of heat about her head ; her legs were slightly swollen. All the arteries sensible to the touch, appeared more dilated than natural : their pulsations were vigorous and accelerated, those of the carotids were very visible, the pulsation of the arch of the aorta occasioned a kind of elevation at the episternal notch. The cubital arteries pulsated violently, and the patient said that she also felt internal pulsation. In constant dread of death, she yielded to despair, and became unconnected in her ideas. The cardiac movements were accelerated, but otherwise exhibited nothing peculiar. All the symptoms became augmented, and death took place on the 27th of the same month amid violent suffering.

Autopsy, twenty-six hours after death. Face tumid ; surface of the body marbled, presenting numerous livid patches of considerable extent. The left pleura contained a small quantity of reddish fluid. Heart larger than natural. Right auricle considerably distended. Nothing particular in either right ventricle or in pulmonary artery. Left auricle normal ; but the left ventricle was, at least, as big again as it should have been, and its walls were sensibly thinned. The

great sinus of the aorta was very developed; the walls of the artery were hard and thickened in many places. The lining membrane was inflamed from its origin to the common iliacs, as also that of the carotid arteries and sigmoid valves.* Independently of this, throughout the entire extent of the vessels, small, white and hard tubercles were observed.

The remaining arteries were sound. Nothing particular was observed with regard to the abdominal organs.

The following is the reasoning of Bertin respecting this case:—'This observation offers us, in all the plenitude of their energy, the principal signs of arterial inflammation. These signs, indeed, consisted essentially in augmented action of the arterial system, in pulsations active, strong, vehement, and vibrating, such as were observed in our patient. The pulsations were so violent at the episternal notch, as to induce suspicion of aneurism of the arch of the aorta.

'Such violence in the arterial dilatations and contractions is the more remarkable, inasmuch as it was associated with thinning and dilatation of the left ventricle, a circumstance unfavourable in a two-fold sense, to the force and vigour of the pulse. We are, consequently, compelled to admit a proper contractile force in the arteries, and an action, to a certain extent, independent of that of the heart.'†

* This appearance was, doubtless, caused by simple imbibition. —J. C.

† *Inflammation de l'Aorte*, p. 35.

In the *Edinburgh Med. Chirurg. Transactions*, vol. i, 1824, Dr. Abercrombie has, with his usual accuracy of detail, described a case of aortic insufficiency, with the *post mortem* appearances; but he has utterly failed in seizing the pathology of the disease.

'To these desultory remarks, I shall only add one case of active aneurism of the left ventricle, accompanied by an appearance which is very uncommon.

'Case 21.—A man, aged about forty, received a severe injury of the left side of the thorax, by a fall from a horse. He soon recovered from the immediate effects of it, but was from that time affected by a train of obscure symptoms in the thorax, which, after a considerable time, began to assume the characters of an affection of the heart. There was obscure deep-seated pain, with occasional attacks of dyspnœa, and a remarkably strong, but regular, pulsation of the whole arterial system, particularly a peculiar and strong jarring of the carotids and subclavians; the pulse generally about 120. The action of the heart was rather stronger and more extended than natural, but by no means corresponding with the remarkable strength of the arterial pulsations, upon which large and repeated blood-letting made almost no impression. His breathing became more and more difficult, with extensive anasarca, and he died about five months after the period of the fall.

Dissection.—The left ventricle was nearly twice the capacity of the right, and in its substance much

thickened, and very firm; the columnæ carneæ were much enlarged. One of the semi-lunar valves presented the appearance of a ring, its body being perforated by an irregular opening, which occupied the greater part of it, and gave an appearance as if it had been torn from its attachment along its base, remaining attached only by the two angles. The other valves were healthy. The right ventricle appeared somewhat enlarged; and the right auricle was nearly twice its natural size, and very thin.

'The prominent symptom in this case was the peculiarly strong pulsation of the arterial system, especially of the large arteries about the neck. The pulsation of the heart did not by any means correspond with it, and, indeed, was much less remarkable than in some of the preceding cases, in which the ventricle was enlarged without thickening. It is also worthy of observation that a strong and extended pulsation may exist without enlargement.'

In the *London Medical Gazette* for the year 1829, Dr. Hodgkin published his observations upon one form of disease of the aortic valves, permitting reflux into the cavity of the left ventricle, and termed by him 'Retroversion of the valves of the aorta'.

These papers are very valuable, more particularly in an anatomical sense. It is the first example of a series of cases brought forward to show the influence of diseased conditions of the aortic valves, admitting reflux, upon the left chamber of the heart.

The anatomical description is given with Hodgkin's characteristic accuracy in every particular. With regard to the constitutional and physical signs, in this early stage of auscultation, much was not to be expected. In one case, however (that of Dr. Cox), one of simple aortic insufficiency, the double murmur was particularly noticed, but without allusion to its site, or explanation given of its cause. So far from this, Hodgkin remarks, 'The contractions of the ventricles were marked by strong impulse, and a constant *bruit de scie*, which presented this peculiarity, that it was double, attending the systole as well as diastole, but not exciting the idea of being at all connected with the auricles. Yet, by a singular coincidence, at the end of this paper, when again adverting to this case, Dr. Hodgkin seems to have divined the physical cause of the double murmur he heard, for he writes, 'It would still appear, that, in the majority of instances there is no *bruit de scie* accompanying retroversion of the valves. The peculiar character of this sound in the case of Dr. Cox is well worthy of attention, as connected with some of the appearances noticed in the inspection. It was repeatedly observed to offer a double or spondaic character, the one part marking the systole, and the other the diastole of the ventricle. The spots of partial thickening on the interior, both of the ventricle and of the aorta, and which had evidently been occasioned by the contact of the elongated and much

* *Edin. Med. and Surg. Journal.* 1832.

retroverted valve, sufficiently prove that the blood on the left side had been subjected to two motions, the one progressive and the other retrograde, in both of which it might easily give rise to some sound as it passed the elongated valve.' Hodgkin states that in these cases the force of the pulse does not correspond to the heart's beat. The violent beating of the carotids, and the thrill of the pulse are, also, recorded. The distinction, too, between the generally undisturbed rhythm of the pulse in aortic compared with mitral valve diseases, is expressly stated. Hodgkin appears to have been taught by his experience that in this particular class of cases, 'depletion seems rather to aggravate than to relieve the distress of the patient'.

These memoirs contain the germs of our present knowledge with regard to both physical signs and treatment. Indeed, had Hodgkin but traced the physical signs with the same success that he did their anatomical cause, the next acute observer had been completely anticipated.

From this date until the year 1832, no additional observations were recorded illustrating the Pathology of *Insufficiency of Aortic Valves.* Throughout the entire historic period of the disease, isolated cases only had occurred, and these so widely scattered through the general literature as to have eluded any attempt to classify, and deduce from them, any general pathologic principles. But, in the year named, a pathologist of eminence, the late Sir D. Corrigan, published his

well-known Memoir. He may be considered, in the strictest sense, the discoverer of the malady; the very first physician to correlate the signs and morbid changes. It is true, that objection might be taken to some of his positions, and, perhaps, from more extended experience, he might himself be disposed to modify some points of his doctrine. I would particularise the assertion that the cardiac impulse is unchanged; again, that collapsing vessels are a *sine quâ non*, as it were, of the disease; and, also, the statement of the non-occurrence of pulmonary congestion. In one other sense I regard Sir D. Corrigan's paper as incomplete, viz.: in not only entirely overlooking the accident of sudden death in these cases, but as even implying an immunity from such event. This contingency, now well ascertained, is most important in a prognostic sense. Still, despite these (except in the last named sense) minor matters, Corrigan's paper is indispensable to the student of aortic disease. Indeed, its value, in a therapeutic sense, is immense. He looked at the disease with a vision clouded neither by authority, nor prejudice, but rather as a physiological pathologist, watching the efforts Nature herself made to mitigate the evil. He was thus enabled to rescue the treatment from blind routine, and to place it on so firm a basis that it never has or could be again abandoned, without manifest disadvantage and even danger.

Dr. Hope* had noticed the 'jerking pulse' in insuffi-

* *Diseases of the Heart*, p. 380. 1839.

ciency of the aortic valves, as early as 1831, and, in the third edition of his work states, moreover, that the murmur in this disease 'is louder and more superficial, opposite to, and above the aortic valves, than about the apex of the heart, by which it is distinguished from a murmur in the auricular valves with the second sound.' Hope's observations connected with the pathology of aortic valvular insufficiency are very brief, but he must have the credit of being the first to indicate the general principle that the murmur was engendered at the immediate site of, and by, the changed valves themselves. This was a great advance in physical diagnosis, inasmuch as murmur, replacing in part, or entirely, the second sound at the base of the heart, and *a fortiori* if of musical character, is, when present, really pathognomonic; the exceptions being so rare that they may, in a practical sense, be wholly disregarded.

On the 6th of June 1834, Dr. Guyot presented to the Faculty of Medicine of Paris his admirable thesis, *On Insufficiency of the Sigmoid Aortic Valves.* He has clearly shown, how considerably the condition of the aortic valves modifies many of the signs enumerated by Corrigan. They may, for example, yield readily to the systolic contraction of the heart, and yet be widely patent during its diastole. In such a case, the phenomena of collapsing vessels would be strongly marked; or, on the other hand, the valves may have the power of motion so limited, consequent upon calcification of

their tissue, that they are almost motionless in the stream, unable to be thrown open sufficiently wide for the outward gush, or to shut close enough to act as a flood-gate to the refluent current. Here, double murmur is met with, showing the conditions alluded to.

Two years later, Dr. Charcelay defended his thesis on the same subject at the Faculty. It is well worthy perusal, and contains much interesting detail connected with the various morbid conditions of the valves in their causal relation to the disease in question. He accepts the physical signs already current.

Almost the last contribution to the literature of aortic insufficiency, superadding any physical sign, is from the pen of Dr. Henderson.* The paper is a most valuable one. It clearly and correctly points out how some of the signs enumerated by Corrigan may be, indeed often are, at times wanting, at all events in the fulness implied in Corrigan's paper. To supply such defect in the symptomatology of the affection, Henderson is of opinion that he has discovered a sign of occasional importance: 'a greatly increased interval between the systole of the heart, and the pulse of the remote arteries, such as the radial'. The question is, has clinical enquiry proved such condition to be essential or only an accident of aortic insufficiency? I believe the latter, and that some degenerative change in the walls of the heart, is additionally present in the cases pre-

* *Edin. Med. and Surgical Journal*, Oct. 1837.

senting such phenomenon. I have examined, carefully, a large number of cases of the disease without discovering the connection. Henderson's statement, that the tortuousness of the arteries becomes more remarkable as the disease continues, requires, so far as I can judge, some qualification. I believe this only happens in cases of marked atheromatous degeneration.

Although, since this period, many esteemed contributions have been made, they have rather been in the way of confirmation than extension of the signs described, and more particularly with reference to the occasional results of the disease.

There is, however, one other addition to the physical signs to be mentioned, and that is the phenomenon of the so-called 'sounding pulse' of Bamberger. He states that, in health, two sounds are heard on auscultation, in the carotid and subclavian arteries, but are lost in those more remote, as the brachial, radial, crural, etc. In insufficiency of the aortic valves, these relations no longer obtain. Not only in the last named arteries, but even in those of still smaller calibre, as those forming the palmar and pedal arches, a sound is audible during their diastole, or, more correctly speaking, a short, almost noiseless shock, nearly resembling that produced by a fillip on the nose. This constitutes a fourth peculiarity of the pulse, very rarely observed under other circumstances, and then only in the larger arteries, as the brachial, crural.

The cause depends upon the stronger tension of the elastic arterial coats, and therefore becomes less distinct as the elasticity of the arteries diminishes, until, in extreme degrees of rigidity, it may wholly disappear. To appreciate this phenomenon clearly, the stethoscope or naked ear must be lightly placed over the site, since pressure generates a hissing murmur without significance, as it is heard even in the normal state. If insufficiency, and particularly the secondary hypertrophy of the heart are not marked, or great debility exist, the sounding character of the pulse may fail'.* This phenomenon must not, I suppose, be confounded with the musical sound, at times, transmitted from actual disease of the valves themselves.

Thus far it has been my endeavour to chronicle the efforts made towards perfecting the pathology and diagnosis of insufficiency of the aortic valves. As happens with each discovery, it was seen, at first, how isolated the cases were, and how long and difficult was the task of generalization. This process was, perhaps, mainly owing to the more rigorous cultivation of morbid anatomy in connection with improved methods of physical research. The motto adopted by *Piorry*, from Hippocrates, was never better exemplified,—'*Pouvoir explorer est une grande partie de l'art.*' The early masters, Vieussens, Lancisi, Albertini, Valsalva, Senac, and Morgagni were conversant

* *Lehrbuch der Krankheiten der Herzens.* 1857.

with inspection and palpation only. By inspection must be understood not merely the amount of visible cardiac impulse, or the phenomena appertaining to the arteries and veins, but also the precordial bulging caused by enlargement of the heart. Under palpation, also, is included both the force of the heart's impulse, and the peculiarity of the arterial beat. Later, however, physical exploration acquired an addition of the greatest value, by the introduction of percussion, by Auenbrugger. The heart could now, within certain limits, be made the subject of actual measurement. 'Speaking of the signs of the enlargement of this organ', he says, 'Signum pathognomonicum hujus mali est, quod locus, ubi cor situm obtinet, percussus in magnâ circumferentiâ, carnis percussæ sonitum exactè referat'.* To the great commentator on Auenbrugger, Corvisart, science owes the detection of another important sign, developed by the heart and arteries, namely, the tactile phenomenon known as *fremitus*, or thrill.† It was, however, the peculiar privilege of Laennec, by the invention of the stethoscope, to give the finishing stroke to the sense-edifice of cardiac diagnosis.

* *Inventum Novum*, etc., 1761.

† It would, perhaps, be more correct to say that Corvisart first gave precision to this sign, as significant of disease of the mitral orifice. Selle (*op. cit.*) had already described it. Speaking of a case he saw in 1782, he writes, 'She suffered unceasingly from such violent palpitation, that each beat of the heart was visible when the chest was exposed, and, on palpation, a kind of oscillation was present, which was attended by *bruissement* (fremitus). I thought an aneurism existed'.

Part II.

PATHOLOGY.

'Has vaticinationes eventus comprobavit.'—Cicero.

The following observations concern some points of the affection known as 'Insufficiency of the Aortic Valves', together with the influence such condition exerts upon the chambers of the heart, and, more particularly, its connection with sudden death—an occasional accident of the disease which merits far greater attention than it has hitherto received.* Indeed, the omission of this accident is almost the only one of importance in the well-known memoir of the late Sir D. Corrigan—a memoir to which we are all so largely indebted.

Before discussing, however, the pathology of the affection, I would invite a reconsideration of the

* The memoirs of Aran and Mauriac should be consulted. The last named author has given a statistic of the cases of death which have occurred in aortic insufficiency; also the later edition of Walshe, *Diseases of the Heart*, where the subject is well discussed. Dr. Walshe states that Chomel, also, was cognizant of the malady.

Professor Peters, in his *Leçons de Clinique*, states that Gendrin pointed out the occasional occurrence of this accident before his pupils, Aran and Mauriac, wrote upon the subject.

Raynaud, *Nouveau Dictionnaire de Médicine et de Chirurgie Pratiques*, tome viii, p. 605, asserts that Briquet made sudden death in aortic insufficiency the subject of a communication to the Society of Medicine of the Seine in 1856.

physiology of the circulation of the blood in the coronary vessels of the heart, inasmuch as our right comprehension of the accident alluded to may greatly depend upon the views held as to the time and manner in which the arterial blood enters the coronary arteries. Upon this point, even in the present as in past time, complete unanimity has not prevailed.

Perhaps, the very large majority believe, with Haller, that the coronary arteries are filled by the heart's systole, and, of course, during the dilatation of the aorta.* But, some few, and I include myself

* According to the statement of the generally scrupulously exact Haller. 'Moreover, in the coronary artery, the pulse is synchronous with the other arteries in the animal body, and the blood, during the contraction of the heart, springs to a greater height.'—*First Lines of Physiology*, translated from the third Latin edition, chapter iv, p. 59. *Vide*, also, some valuable remarks of Portal, respecting the position of the openings of the coronary arteries with respect to the valves.—*Anatomie Médicale*, tome iii, p. 147, Paris, 1803. Luschka, *Anatomie des Menschen, erster Band*, s. 402, Tubingen, 1863, after stating his own opinion that the openings of the coronary arteries were sometimes above, at other times below the free border of the valves, adds, 'Many earlier observers were aware that the openings of the coronary arteries were sometimes covered by the semi-lunar valves, at others above them.' Morgagni, who had given particular attention to the matter, found that out of eighteen bodies in which the openings were sought for, in only five instances were the openings covered, in thirteen they were visible above the valves. Morgagni asserted, in defending his thesis against Fantoni, that the openings of the coronary arteries, whatever might be their relative position, could not be closed during ventricular systole. The dispute has been somewhat warmly renewed in recent times by Hyrtl and Brücke. For a good account, both of the old and modern controversy, consult especially, Henle, *Handbuch der Systematischen Anatomie des Menschen*, dritter Band, erste Abtheilung, Gefässlehre, s. 86,

among the number, think it not improbable that the arteries may be filled by the systole of the aorta, and, consequently, during a period corresponding to the heart's diastole. Others, to reconcile the difficulty, have suggested that the arteries receive blood during the acts of both systole and diastole.

The chief arguments against the injection of the arteries during cardiac systole, may be thus stated:— first, the retrograde course taken, and the angle formed by the arteries: second, the position of these vessels in relation to the sigmoid valves; and last, the apparent want of adaptation of the time, as respects the nutrition of the cardiac muscle. There is experimental proof that, during the period of systole no blood enters the parenchyma of the organ; the observations of Harvey, showing that, in some animals, during this act, the cardiac wall, while it is most dense, is at the same time most pallid: semi-transparent, and only darkens during diastole, when the tissue is again pervious to the blood.*

Braunschweig, 1868. More recently, Ceradini has upheld Haller's view, on physiological grounds. I find myself in error in stating that Boerhaave suggested the idea of the arteries being injected during aortic systole. Fantoni, 1652-92, appears to have first advanced the doctrine on physiological grounds. According to Wunderlich, *Geschichte der Medicin*, s. 135. 1859, Fantoni also pointed out the connection between hypertrophy of the heart, valvular diseases, and aortic aneurisms, together with the symptoms they manifested during life.

For the general state of opinion on these points at the time of the early masters, who, by the way, paid quite as much attention to them as ourselves, consult Senac, *op. cit.*, liv. i, ch. v, tome i.

* Unless Harvey restricted his observation to the translucent

It is, I think, no valid objection to this view, that Haller should have observed blood forced out of the arteries during systole, inasmuch as this act would naturally give rise to reflux of a given portion of blood from the interstitial branches into the primary trunks, and thus explain the phenomenon witnessed

hearts of inferior animals, it is evident he meant that not only the ventricles, but also their walls, were partially emptied during systolic contraction. 'Ex his mihi videbatur manifestum, motum cordis esse tensionem quandam ex omni parte et secundum ductum omnium fibrarum, et constrictionem undique; quoniam erigi, vigorari, minorari, et durescere in omni motu videtur: ipsiusque motum esse, qualem musculorum, dum contractio fit secundum partium nervosarum et fibrarum. Musculi enim, cum moventur et in actu sunt, vigorantur, tenduntur, ex mollibus duri fiunt, attolluntur, incrassantur: et similiter cor.

'Ex quibus observatis rationi consentaneum est, cor, eo quo movetur tempore, et undique constringi, et secundum parietes incrassescere, secundum ventriculos coarctari, et contentum sanguinem protrudere; quod ex quarta observatione satis patet; cum in ipsa tensione sua, propterea quod sanguinem in se prius contentum expresserit, albescit; et denuo in laxatione et quiete, subingrediente de novo sanguine in ventriculum, redit color purpureus et sanguineus cordi.'—*De motu cordis et sanguinis animalibus.* Cap. 2. *Ex vivorum dissectione, qualis sit cordis motus.*

The late Dr. Sibson, as is well known, devoted much time and attention to determine the order of succession of the movements of the heart, as observed in some of the larger mammals. The results, even to the minutest details, were recorded with an accuracy, fulness, and truthfulness, to be appreciated only by those who knew him. His observations mainly coincide with those of Harvey. 'The appearance of the heart in motion is very striking. The ventricles during their systole contract from all sides upon their own centres and become wrinkled, and the arteries and veins on their surface are full and tortuous, while the auricles become purple, plump, and glistening. During the diastole, the aspect is reversed; the ventricles enlarge and become smooth, their superficial vessels almost disappearing, while the auricles shrink and become pale and wrinkled.'—'Movements of the Heart', Reynolds' *System of Medicine,* vol. iv, p. 65.

by this great observer upon section of these vessels. It is clear, that the tissue is rendered pallid from the reflux of the arterial, and onward passage of the venous blood.

The facts favouring the hypothesis, that the coronary arteries may be filled during the systole of the aorta and dilatation of the heart, are: first, the unique course and condition of the coronary arteries, placed with their mouths in direct contiguity to the aortic flooring, and, in their course from the aorta, taking a direction easily to be injected by the blood after its shock against, and rebound from, the closed sigmoid valves. It is, also, during the relaxed condition of the cardiac walls, that the interstitial vessels are most readily filled, and thus, probably, the subsequent act of contraction, in part at least, determined.* Experiment additionally testifies that the coronary arteries, when injected with blood, immediately stimulate the heart to contraction.†

It would certainly be unsafe to draw rigorous conclusions from such coarse experiments as we are permitted to institute on the dead subject, with regard to the systolic injection of the coronary arteries: the process of ventricular contraction is so essentially vital. But we can, with far less risk of error, ex-

* *Vide Contributions to Cardiac Pathology*, by the Author, p. 4.

† It is not from any single fact, but from the collocation of many, that the argument gains in point of probability. One series of phenomena, apparently trivial, when in juxtaposition with others, not unfrequently assumes a high degree of importance.

perimentalize upon the aorta and its valves. Here the mechanism, aiding the general circulation, is of an almost purely physical character. When the blood tension is greatest on the ventricular wall, the pressure tends to raise the aortic valves. When, on the contrary, the tension preponderates on the side of the arterial walls, the valves are closed. If the aorta and its valves are healthy, and freed from clot, and the coronary arteries dissected and divided, water, poured in the common trunk and submitted to gentle pressure downwards, most readily passes by the divided coronary vessels.

In further support of this position, it was found by the philosopher Bernouilli, that if fluid was injected into a tube of given calibre, it would pass with facility into secondary tubes, the direction of which was in harmony with that of the main branch; but if, on the contrary, the direction of the secondary tubes was opposed to that of the trunk, not only would the onward current be prevented passing therein, but that fluid would actually be drawn up into the latter from a reservoir below.

It now remains to be shown how far the view I advocate can be made to harmonise, in some essential particulars, with the pathology of the disease in question.

The ventricles of the heart, in common with every other organ of the body, require some respite, however short, from active labour. Such respite is

afforded by the semilunar valves, which, by a mechanism purely physical, resist the downward pressure of the columns of blood completed by the last contraction of the ventricle. Thus is the heart, as it were, momentarily relieved of the principal portion of its burden. In a healthy, physiological condition, such relation is maintained between the power of the ventricles and the resistance to be overcome, that they are enabled at their next contraction to force these flood-gates barred by backward blood, and thus to maintain the balance of the greater and lesser circuits. But should these gates refuse to open, or become too narrow ever again to close, then the heart, baffled by the unusual, though intermitting effort, or exhausted by its necessary constancy, may gradually sustain such injury, both in its vital and physical endowments, as is no longer compatible with health or even with life. It is, however, more particularly in reference to insufficient closure that the present remarks are intended to apply, and it is my aim to show, moreover, how such faultiness in the valvular mechanism may furnish the conditions under which sudden death, by cardiac paralysis, occasionally occurs.

It will be found both practical and convenient to divide insufficiency of the aortic valves into three stages:—the Incipient, or irritative; the Physiological, or compensative; and the Degenerative or failure stage, inasmuch as it is the ventricle which plays the leading part in, at least, the two latter

stages of the affection. The first stage, when the direct result of rheumatic valvular aortitis, so far as I have observed, appears to be one simply characterised by the signs of general cardiac excitement, and in no wise susceptible of physical differential diagnosis from mitral disease, or even nascent pericarditis.* Increased, often irregular impulse, obscured sounds, and, later, slight systolic murmur, are alone observed. It is true that in pericarditis, when either a friction or leather-creak sound is audible, or the percussion area becomes suddenly and greatly enlarged, there could be little chance of diagnostic error. But it is found, clinically, that pericarditis in its developmental stage, occurs at times without the stethoscopic signs, and certainly without any greater extent of dulness than would consort with that caused by a heart enlarged by distension of its cavity and congestion of its walls. The second stage is, as is well known, characterised by the striking phenomena of greatly increased impulse of the heart, and throbbing or collapsing of the superficial arteries. Here, the physiological hyper-

* In this stage, if endocarditis is the originating cause, the valves are *tumid* from subserous effusion. This condition, while muffling the sounds, may really prove for a time an additional barrier to regurgitation. In fact, the irritative stage is one characterised rather by the phenomena of obstruction than by those of regurgitation. Dr. Richardson's experiments in this particular are most interesting. It is in a subsequent stage, when the plastic matter unabsorbed is gradually undergoing contraction, that the phenomena of regurgitation become as gradually developed. Regurgitation may also occur from vegetations on the free edges.

trophy of the chamber, consisting of a real addition of healthy, contractile tissue, is a genuine compensation—an instinctive accommodation of Nature, under injury sustained. Let us consider how impossible it would be for the circulation to continue if the strength of the left ventricle were unchanged and any great amount of reflux occurred. For the requirements of the economy there must be as much blood sent into the aorta, *plus* the quantity which reflows. To make this provision, is the final cause of the hypertrophy of the left chamber. The amount of collapse of the vessels, practically speaking, measures, as I believe, the extent of patency of the aortic valves. This second stage may, at times, persist for years without passing its original limits.

It is in the early period of the third, or degenerative stage, that we are to anticipate the supervention of the accidents or complications of the affection. I wish, here, merely to allude to such as involve the lungs, right heart, and aortic trunk. There are two modes by which the lungs and right ventricle become implicated in this affection. One, almost purely mechanical; the other, vital. The former acts in the following manner. When the dilated hypertrophy of the left ventricle has attained such magnitude as to merit the epithet of *cor bovinum*, it occasionally encroaches so much upon the area of the left lung, as to cause considerable condensation of its tissue.* Such

* Rosenstein holds the same view. Ziemssen's *Handbuch*, sechster Band, s. 99, 1876.

condensation, by obstructing the pulmonary circulation, tends directly to the production of dyspnœa, catarrh, hæmoptysis, and almost necessarily, as in mitral disease, to dilatation of the right side of the heart.

To the second, or vital mode I shall shortly allude.

The aortic trunk, also, sustains direct injury. The constant over-force of the ventricle tends, first, to the dilatation of this discharging tube, and eventually, at times, to its entire loss of elasticity, and atheromatous degeneration.

These conditions, in their turn, react injuriously upon the chamber. The impaired elasticity, by obstructing the onward current, furnishes its contingent to the hypertrophy, and, in the degenerative stage, weighs heavily on the failing power of the heart. Morbid anatomy has recently brought to light the strict correlation between hypertrophy of a ventricle, and atheroma of the trunk in immediate connection with it. Even in hypertrophy of the right ventricle, it is frequently found that the pulmonary artery has undergone atheromatous degeneration.

Such change in the aorta very often extends to the mouth of the coronary arteries, modifying the nutrition of the cardiac walls, and tending to still further results.* I cannot too emphatically redirect attention to the diseased condition of the aorta and destruction of its elasticity, as an almost invariable sequence of disease of the aortic valves.†

* *Vide* Dr. Adams, *Dublin Hospital Reports.*

† If the natural supply of blood is cut off from a muscular

This undeniable fact I regard of cardinal importance, inasmuch as such incapacity of reaction of the aorta must, if the view I advocate of the time of injection of the coronary arteries be correct, prevent either a constant or sufficient repletion of these nutrient vessels, and thus directly favour cardiac mischief. Here, I apprehend, we but trace the operation of a recognised law in pathology—that in proportion as an organ is over-worked and under-fed, degeneration of structure is sooner or later of almost inevitable necessity.

No affection could more strikingly exemplify the fatal epigraph of Corvisart — *hæret lateri lethalis arundo.* From the coustant overstrain of the ventricle; the perverted nutrition of the cardiac walls; and from the cachexia, general and local, induced by disturbed balance of the circulatiou, the left veutricle, eventually, undergoes an irreparable change of a fibroid or, more commonly, of a fatty nature. The extreme limit of the third, or degenerative stage, is then attained.* The ventricle, no longer capable of efficient contraction, still further yields from the blood-pressure on its walls, and secondary incompetency of the mitral valve occurs. This enlarged capacity of the left chamber must never be lost sight of in insuffi-

organ, paralysis will as speedily and certainly follow, as if all communication with the brain were interrupted.

* Degeneration of the tissue, whether fibroid or fatty, when extensive, must equally involve both capillaries and nerves.

ciency of the aortic valves. It is here that it nearly attains its *maximum*. It is the direct cause of the cyanotic signs, and, remotely, furnishes a condition for sudden death. The marked throbbing, too, of the heart and arteries gradually lessens, only to be reproduced at intervals. This fact is often noticed even by the non-professional observers of the case.

In consequence of the incompetency of the mitral valve, just named, being superadded to the diseased walls, there is a transference of the objective signs from the left to the right side of the heart, and now all those phenomena, at times, gradually appear, common to the closing stage of heart disease. This is, perhaps, the more ordinary clinical history. But there remains the consideration of the mechanism of those accidents, so to speak, by which life is, occasionally, suddenly cut short. Leaving among the outstanding points, death by embolia, or plug detached from the parts immediately diseased, sudden death in aortic insufficiency is almost invariably syncopal, or blended with more or less of asphyxia. Two causes exist for the occurrence of this accident; the one of extrinsic, the other of intrinsic origin. The extrinsic cause is sudden pericardial effusion, induced by the cardiac venous congestion from the diminished *vis a tergo*, owing to insufficient blood tension of the coronary arteries. The intrinsic cause, I assume to be as follows, although it is difficult to state the precise time or invariable exciting cause. It may be mental shock,

bodily pain, effort, or general exhaustion. But, granting the degeneration of the enormously dilated left ventricle, whenever, from the influence of these exciting causes, singly or concurrently, the exhausted heart permits the diastole to be prolonged, and the *hiatus* at the aortic mouth is sufficiently large, the systemic blood flows unimpeded into the left chamber, which acts as a vast reservoir.* Thus is the general system, and, particularly the brain, robbed of the needful stimulus. The *point d'appui* for the backward blood being lost, and the elasticity of the aorta gone, the coronary arteries, on the theory given, remain unfilled. Add to this the disposition to paralysis of the left ventricle from the enormous blood-pressure on its walls, and we have the sum of the conditions under which death occurs by what is known as cardiac syncope.

The physical signs of aortic insufficiency are so well known, and, for the most part, so readily detected, that detail is superfluous. It is only with regard to the significance of one or two of them that I would ask the reader's consideration. First, it is matter of clinical experience that, occasionally, in cases of defective aortic valves, the first sound of the heart is absent, although the cusps of the mitral valve are healthy.

* Vesalius records an instance in which the left ventricle contained two pounds of blood.

Cruveilhier,* and, later, Leared,† held that the first cardiac sound (arterial, it should have been said, of Skoda‡) was essentially a fluid sound caused by the

* 'Ectopia Cordis', *Gaz. Méd.*, No. 32, 1841.

† Sounds caused by circulation of the blood.

‡ For much valuable information respecting the sounds of the heart, consult Gerhardt: *Lehrbuch der Auscultation und Percussion*, 2te Auflage, s. 187-97 inclusive. Paul Niemeyer, in his erudite monograph, *Handbuch der theoretischen und klinischen Percussion und Auscultation*, Band ii, s. 75, Erlangen, 1870, thus summarises Skoda's view—'Es ergeben sich also aus dieser Analyse acht einzelne Töne, welche unter normalen Verhältnissen bei dem Isochronismus des Klappenschlusses und der Contraction in beiden Hälften als je 4 Paare hörbar, welche aber unter abnormen Verhältnissen isolirt zu auscultiren sind.' The original work, however, is indispensable to those who desire to go thoroughly into the entire subject. In the supplement to his first work (*Grundriss der Percussion und Auscultation*, Erlangen, 1871), Niemeyer expressly states in the preface, 'Complicirte Fragen, wie Herzschlag und Herztöne, sind nicht apodictisch entschieden, sondern eklectisch auseinandergesezt.'

Much to the same effect writes Woillez (*Traité Théorique et Clinique de Percussion et d'Auscultation*, Paris, 1879), one of the latest French authors on auscultation—'Malheureusement, malgré l'importance et la multiplicité de ces publications, beaucoup de questions relatives à l'auscultation du cœur restent encore obscure.' One pair of sounds seems, by pretty general consent, to have been omitted from Skoda's list. The second sound, heard over the body and apex of the ventricles, being regarded as the transmitted sound of the arterial semilunar valves. But, I quite believe that there are residual cases that admit of explanation on no other theory.

Sibson watched the action of the mitral and tricuspid valves exposed by dissection. His description, I think, gives physiological support to the views of Skoda and Julius Roger as to the probability of a second ventricular sound caused by rapid disengagement of the valves. 'In order that I might see the movements of the mitral and tricuspid valves, I cut out the heart when beating vigorously, and immersed it in water. The ventricles

impact of the blood columns in the ventricles and arterial outlets respectively, the valves being in their physiological condition.

Consequently, in aortic insufficiency, where the valves are diseased, the first normal sound gives place to murmur. Undeniably true, however, though it be that, in the majority of such cases, the first sound ceases to be audible over the body and apex of the left ventricle, it might be simply masked by the in-

contracted with force, and expelled the water from the great arteries during each systole. The jet from the aorta was six inches in length. The segments of the mitral and tricuspid valves were seen to come together at their notched and bead-like margins, so as to close the valves during the systole, and prevent the efflux of a drop of liquid. At the beginning of each diastole, the margins of the valves separated quickly from each other, so as to admit the flow of water freely into the cavity.'—*Op. cit.*, p. 68. Unless I mistake, there may be found, occasionally, equal support from clinical observation.

In a most remarkable case of cardiac disease, probably congenital, an opinion strengthened by the addition of two lobulettes to the left ear, exhibited by me at the Medical Society of London, and described in the current volume of *Transactions*, there unquestionably exists imperfection both of the aortic and pulmonic outlets. The heart is felt distinctly beating across the chest, from beyond the left axillary, to the right axillary line. A second sound is most distinctly audible. How is it originated? I exhibited another case a few evenings ago, before the same Society. The patient was a young woman, suffering from mitral constrictive disease, the result apparently of rheumatic fever. One day, upon examining the heart, a well-marked presystolic, followed by systolic, murmur is heard; another day, the presystolic murmur has ceased, and a long continued diastolic murmur, of comparatively pure character, succeeds the systolic murmur. A day or two later, following the systolic murmur, a triple quasi-reduplication is heard, evidently the result of a divided murmur, as Skoda has shown. So that, in this case, three different observers, examining the case on different days, would arrive at the most opposite conclusions respecting the physical signs.

tensity of the murmur, since, occasionally, in cases of aortic insufficiency, where murmurs of but moderate intensity exist, upon ausculting far to the left of the aortic area, and far away from the *focus* of murmur, the first sound is heard. The above view is therefore too exclusive. One more comprehensive is required to account for its occasional extinction. Kürschner* states, 'If the arteries are cut away from a heart while beating (*in situ*), the first sound is still heard; the second ceases at once. In a heart also, removed entirely, the first sound, but never the second, is audible'.

I would direct very particular attention to the experiments performed upon large dogs, by my friend and former colleague at the Grosvenor Place School of Medicine, Professor Halford†, now of the University of Melbourne. These experiments were often repeated, and the action and sounds of the heart observed by the hour together. Upon clamping the superior and inferior cavæ with bull-dog forceps, and compressing the pulmonary veins with the thumb and finger, the action of the heart continued, but the sounds instantly ceased, and as instantly became again audible on re-admitting blood into the chambers. There could be no possible mistake in the matter, inasmuch as the experiments were witnessed, verified, and repeated, separately, at different times by some of the most skilled and able observers of the day.

* *Herzthätigkeit*, s. 101. Wagner's *Handwörterbuch der Physiologie*, 1844.

† *The Action and Sounds of the Heart*, 1860.

The Ludwig-Dogiel experiment, supported by Bayer, went to prove that the first sound (muscular) was still audible in the bloodless heart. But, as was subsequently shown by Guttmann and Rosenthal*, although a sound was heard under the observed conditions, it was of an entirely different character to that of the first normal sound; and by comparing both, in hearts emptied of blood, and those through which blood was passing, valvular tension was found to be the principal cause; muscular contraction being quite subordinate.

The blood, states Kürschner†, is the stimulus which excites movement during the normal condition of life. This fluid may act either by its chemical qualities, or it may excite the action of the heart in a mechanical manner. This statement is abundantly confirmed by the experiments of Haller and others.‡

Bamberger has attempted to explain how, in cases of aortic insufficiency, the first sound loses its simply valvular element. But this is not extinction of sound. Now what explanation is to be given of a fact beyond dispute, accepting the more common theory that the

* Niemeyer, *op. cit.*, Band ii, i Abtheilung, s. 90; also, Guttmann, *Handbook of Physical Diagnosis*, English translation, p. 265, 1879.

† *Herzthätigkeit*, s. 78. Wagner's *Handwörterbuch der Physiologie*, 1844.

‡ Refer particularly to O. Lannelongue, *Circulation veineuse des parois auriculaires du Cœur* (Thèse de Paris, 1867), quoted by Luton, article 'Cœur', *Nouveau Dictionnaire de Médicine et de Chirurgie Pratiques*, tome viii, p. 309. Luton's article will well repay careful study in its entirety.

first cardiac sound is caused by the closure of the auriculo-ventricular valves? What can be the relation subsisting between a defective aortic flooring and the extinction of the first sound of the heart? To my mind the view of Traube and Fräntzel* commends itself as being more in accordance with current teaching as to the site of the first normal cardiac sound.

'In cases of well-marked insufficiency of the aortic valves, I have', says Traube, 'frequently found a more or less decided flattening and elongation of the papillary muscles of the left ventricle, without any appreciable alteration of their volume. This condition stands in singular contrast with the marked hypertrophy which the dilated ventricle undergoes; and it is the more remarkable as, in cases of dilatation and hypertrophy of the left ventricle, without affection of the aortic valves, the papillary muscles are, as usual, round and correspondingly hypertrophous. Upon a transverse section of these muscles, there is, almost constantly, observed a number of irregular white *striæ*, within which, as is shown by microscopic observation, the muscular structure is replaced by fibrous tissue. In the majority of instances there is no warranty for the notion of inflammatory exudation, since neither in the endocardium of the degenerated papillary muscles, nor on the wall of the ventricle, is such fibrous tissue observed. The change, consequently, appears to be essentially different from that of ordi-

* 'Ueber zwei eigenthümliche Phänomene bei Insufficienz der Aortenklappen.' *Berlin Klin. Wochen.*, 1867.

nary inflammation. The elongation and flattening of the papillary muscles, indicate, therefore, that these structures were exposed to a permanent *extension* in the direction of their vertical, and to a permanent abnormal *pressure* in the direction of their transverse diameters. Indeed, from observation of the phenomena occurring in cases of marked insufficiency of the aortic valves, during the period of diastole, in the left ventricle, the conditions are discerned which have thus affected the papillary muscles. In such cases, the relaxed ventricle is supplied from a double source. It receives, simultaneously, blood both from the left auricle and from the aorta. As a consequence of this abnormally large addition, its walls must soon acquire a tension greater than the pressure under which the blood flows from the auricle. A current is consequently originated with a direction from the ventricle towards the auricle, and which effects the closure of the mitral valve.

'The mitral valve closed, the contents of the ventricle thus cut off from the auricle, are submitted for a period to the pressure equal to that which presses upon the blood in the aorta. The blood from this latter tube would continue to be poured into the ventricle to distend it further, until finally the tension of its walls is sufficiently great to maintain the balance of the pressure in the aortic system, or at least, until the moment preceding the ensuing systole. That this *series* of results do actually follow, two facts concur to prove. One is, that in insufficiency of the aortic

valves, despite the integrity of the mitral valve, the systolic sound at the apex so frequently fails; and the other, certainly a rare one, is, that in insufficiency of the aortic valves a loud diastolic sound is audible at the heart's apex, but at no other point of the cardiac region. The condition of the papillary muscles, during these phenomena, admits of the following explanation. From the moment that the mitral valve is closed, their points of insertion into the cardiac wall, in consequence of the increasing distension and tension of the ventricle, become, continually, further removed from the point at which their tendons unite with the curtain of the valves; whilst the bellies of these muscles become more and more strongly pressed. If we reflect, on the one hand, upon the slight comparative elasticity of the living muscle, and, on the other, on the unusual amount of the forces acting upon it, it is obvious that the frequent recurrence of these actions for years with each diastole must, finally, produce permanent disturbance of the sarcous elements from their natural equilibrium, and, consequently, a permanent elongation and flattening of the belly of the muscle.

'But it is not less important to know that, under such conditions, the nutrition of the muscles also undergoes a change.

'The molecular interchange between the contents of the capillaries and the cardiac muscular tissue, and, as matter of course, in those portions of it which constitute the papillary muscles, occurs, almost exclusively, during diastole, since, during contraction of the

muscle, the contents of the capillaries must be almost exclusively forced into the veins. But under the influence of the unusual pressure to which the papillary muscles are subjected, in cases of insufficient aortic valves, the repletion of the capillary vessels cannot be normal, even during diastole; they must, in fact, be in part narrowed and in part pressed, even to the annihilation of their area. The necessary consequence of this condition is, as may be imagined, a disturbance of molecular interchange, in other words, a faulty nutrition of the muscular tissue. That muscular, under abnormal conditions, can immediately degenerate into fibroid tissue, is proved beyond doubt in the recent work of *Billroth*.'

But, while the closure sound of the mitral valve may, even in a physiological condition of this structure, become extinct from the cause assigned by Traube, we must not forget that in aortic insufficiency, the first sound of the heart may also become modified from a direct pathological cause: that is, by the continuous extension of the atheromatous process from the aorta to one curtain of the mitral valve; a fact explained by the descriptive anatomy of the structures.

I would next venture to criticise the ordinary teaching, that the differential diagnosis of aortic regurgitant murmur may be, at least in part, established by the clear click of the valves of the pulmonary artery, auscultated to the left of the sternum. I am far from

asserting that this precept is not occasionally correct; but, so far as my opportunities of observation extend, I should, in the majority of cases, deny the validity of this proposition. When the diastolic murmur is at all intense, I believe that the sound of the pulmonary artery is always more or less obscured, and, in the case of musical murmur, *a fortiori*, completely so. Indeed, if we bear in mind the anatomical condition existant, and consider how readily vibrations are excited in contiguous and homogeneous structures, the difficulty of conceiving the reverse is obvious. The last point for question, relates to the significance of a simple diastolic murmur, if at all well marked, at, or even a little beyond, the left apex. Such point has before been hinted at in Traube's remarks, and I have, on more than one occasion, after death verified its connection with simple aortic disease. Such a murmur may practically (for the exceptions are so few) be regarded as the physical expression of aortic insufficiency, and this, the more certainly, if percussion signs of enlargement of the right ventricle and general venous stasis, are absent.

But, with regard to the semeiotic importance of simple murmur, whether of limited or of wider range, we must be cautious not to allow it to transcend its just value. I would venture the opinion that such murmur indicates the kind, but not the degree of the lesion. This latter condition is measured, as before stated, by the amount of collapse of the vessels.

Moderate obstruction at the aortic mouth is to be considered a most important kind of compensation for aortic insufficiency. An immobile, or still healthy valve, mingling its proper sound with the murmur, arrests and breaks the refluent stream, and thus directly tends to prevent collapse, as also that worst evil of the disease—dilatation.

It is not, perhaps, saying too much, that the management of these cases is not unfrequently, even now, matter of mere routine.

To treat a diseased heart, not only with ordinary success, but even with ordinary safety, it is not enough that the practitioner be thoroughly conversant with the workings of Nature to compensate for injury done to its valvular apparatus, or even, when her resources are exhausted, with the simultaneous or successive steps that lead to death. Perhaps the larger and more valuable part of his knowledge is derived from the clinical study of the congestion of the heart, as also of the congestion and the vicarious action of the other large organs;—now acting as safety valves by increased secretion; and now again, as a consequence of such congestion being inadequate to the task of eliminating refuse material, thus giving rise to a chronic asphyxiating process, and eventually entailing degeneration of their proper structures. The laws, too, of the reciprocal influence of the various organs, are of the greatest importance to a right

appreciation of the use of remedial agents. It is astonishing in cases of long-standing disease of the valves associated with a weakened heart, how slight a cause will terminate life. Ordinary catarrh, congestion of the coronary veins, may determine respectively pulmonary œdema or effusion into the pericardium, and it requires but a small quantity of such effusion to exhaust the already weakened heart.

In the incipient stage of aortic valvular disease, the most important question that therapeutics can ask of pathology, is that which regards the causation of the affection. If this is the direct result of rheumatic fever, those agents are, of course, indicated which experience has invested with a more or less specific action over the rheumatic poison, and subsequently the long continued use of moderate counterirritation, with the internal use of iodide of potassium, alone or in combination, with a view to the absorption of the exudation matter. Simultaneously with these means, the avoidance of the ordinary causes of cardiac excitement is to be sedulously cared for. It is certain that valvular disease of the aorta, occurring as stated, is amenable to treatment and occasionally so far modified as to leave no signs of either general or local disorder. And the same measures would be more or less applicable to cases of strain of the valves. On the other hand, when atheroma is probably causative of the affection, treatment is, for the most part, abortive.

It is ever to be remembered that, in this incipient stage, the phenomena of cardiac excitement are, for the most part, but the reflex of the obstructed blood current. So that here we treat the valve disease, *qua* valve; while, in the later stages, with permanent valvular obstruction, it is to the chambers of the heart that our remedial measures are to be addressed.

Should the means employed fail to achieve the end proposed, the valves may ultimately remain permanently patent, admitting of a greater or less amount of regurgitation into the left ventricle. In most cases, however, such an amount of hypertrophy quickly follows the dilatation of the ventricle, as fairly to maintain the balance of the circulation.*

In this second, or compensative stage, one most frequently presented to our notice, and characterised by so many striking phenomena, all medical interference is, strictly speaking, absolutely contraindicated.† This stage may, and often does, endure for years with but little appreciable encroachment on the vital powers (even permitting a life of active exertion), unless any of the accidents of the affection, induced by some excess, should suddenly supervene. It may, however, happen, even in these cases, that owing to impaired health, the adjustment temporarily fails without the degenerative stage supervening.

Here, in cases where the dilatation is greatly in excess of the hypertrophy, the mitral valve may, for

* *Vide* Rosenstein, *op. cit.*, s. 95.

† *Contributions to Cardiac Pathology*, p. 80.

the time, yield from secondary insufficiency, and modify both constitutional and physical signs. It is under such circumstances that perfect rest, warmth, nutritious diet, ferruginous tonics combined with digitalis, render such pre-eminent service.*

In cases attended (as so often happens) with marked anæmia, which greatly intensifies both physical signs and constitutional symptoms, and, if long continued, may probably cause, or, at least, accelerate the degenerative stage, the mild and easily assimilable preparations of iron here attain their maximum of value.

It is in the degenerative or failure stage, that subjective phenomena acquire special prominence, and, in like proportion, our means of relief become narrowed. There is (unless life be suddenly cut short) a gradual though steady advance towards asthenia from general implication of the chambers of the heart. Occasional angina-like pains, common to all the stages of aortic insufficiency, are now so frequent, and the paroxysms so severe, especially by night, as to form part of the natural history of the stage. They must

* To estimate the comparative value of treatment in cases with or without actual degeneration of cardiac tissue, let us suppose a simple though advanced stage of mitral disease. Here we may have effusion, oppression, and general congestion caused by yielding of the walls of the right side of the heart, again and again relieved by properly directed remedial measures—digitalis heading the list. This agent does its best when, as Traube has shown, by increasing blood-pressure in the aortic system, it quickens the renal secretion. And all this because no actual change of structure of the wall of the heart has for the time occurred. How widely different the result in the opposite condition.

not always be regarded as dependent on simple neuralgia of the cardiac plexus, or other nerves, inasmuch as Professor Peters* has conclusively shown that actual neuritis and disorganisation of the plexus is frequently found, either with or without concomitant inflammation of the phrenic nerves.

It is scarcely necessary to allude to the remedies in ordinary use, further than to observe that inhalation of 4 or 5 minims of nitrite of amyl is the more generally and immediately serviceable. If this remedy cannot be borne, 15 or 20 minims of chloroform may be substituted on each paroxysmal accession; or the hypodermic injection of $\frac{1}{6}$th of a grain of morphia. Now and then, half a drachm of Hoffmann's anodyne in camphor mixture acts well and quickly, especially when, as so constantly occurs, the stomach is distended by flatus. But, I think, where the paroxysms though frequent, are not of extreme severity, that occasional benefit may result from the exhibition of oxide of zinc in as large doses as can be tolerated. The remedy may be administered either in milk or in some aromatic water with mucilage, and, if nausea result, with the addition of 2 or 3 minims of dilute hydrocyanic acid. In addition to the zinc, from $\frac{1}{4}$ to $\frac{1}{2}$ a grain of extract of aconite may be given night and morning. Blisters over the sternum, from time to time, frequently give marked relief.

Our further care should simply be to enforce, so far as is practicable, the avoidance of all the mental

* *Leçons de Clinique Médicale*, 2eme edit., tome i, p. 441, *et seq.*

and physical influences likely to embarrass the heart's action, and by tonics and the diffusible stimulants to try and rally its failing efforts.

Attention to diet is of paramount importance. There is no cause of cardiac suffering, or even of death, more common than dietetic imprudence in the organic diseases of the heart.*

In this stage, digitalis, at times that true opiate of the heart, as Bouillaud terms it, and often so indispensable in disease of the mitral valve, both for the purpose of equalising and steadying the heart's contractions, and for prolonging the period of diastole, to allow sufficient blood gradually to flow into the left chamber for the requirements of the general system, is, in aortic insufficiency of questionable efficacy;† in-

* As a rule the continuous administration of alcoholic stimulants, unless in moderation, is of doubtful efficacy; otherwise, they may oppress rather than give tone to a weakened heart; in consequence, also, of the impaired action of the eliminating organs, they may further interfere with the already compromised function of hæmatosis.

† Under this latter condition the use of full doses of digitalis is not only sanctioned, but strongly recommended, with the direct view of increasing the tonic contraction of the heart. I am free to confess that, until a much wider experience is brought to bear upon this reputed efficacy of digitalis in this particular stage, I have a distrust of the agent—a distrust, unluckily, not merely theoretical. We cannot at all times ensure its tonic, nor quickly enough arrest its untoward action. Admitting the possibility of the former action over portions of undegenerate fibre, while a considerable remainder has undergone conversion into fat, there might be risk of a solution of continuity. This is an objection already taken by a very original thinker, and, at least, deserves consideration. Besides, how does a fatty ventricle rupture at times, unless under a more or less momentary increase of tonic contraction?

asmuch as to incur any risk of prolonging diastole, is to favour the worst result of aortic insufficiency—further dilatation of the systemic ventricle which has already lost effective contractile power.

It has been already pointed out how in this advanced stage the characteristic throbbing of the heart and arteries gradually lessens. We would now gladly restore this apparently over-action of the left heart, which formed the great barrier to the oppression and effusions that now threaten. But, it is precisely at this juncture—amid all these accidents—where remedies are most wanted, they are least to be found. It is true that, if life were imperilled by the imminence of cardiac syncope, we might perchance, if present, successfully interfere, by placing the patient recumbent; by the use of external and internal excitants; and, beyond all, by the sudden application of cold to the region of the heart. But, under the more ordinary circumstances, however, the remedies in use, as iron, zinc, or strychnine, must fail, for they cannot rechange fibroid or fatty tissue into healthy muscle. Science knows no agent that can restore vital contractility to a structure which has lost from disease its originally inherent power; more especially to one so immediately essential to the maintenance of life as that which constructs the left ventricle of the heart.

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BY THE SAME AUTHOR.

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